probably activates mechanisms within the collateral network to enhance spinal cord blood flow, and thereby minimizes ischemic cord injury after subsequent extensive SA sacrifice. After coiling, extensive single-stage TAAA repairs in patients who are unable to undergo conventional 2-stage procedures could be performed at a much lower risk for spinal cord injury. A clinical trial in a population at high risk for postoperative paraplegia may be appropriate.

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Discussion

Dr Joseph S. Coselli *(Houston, Tex)*. Thank you to the AATS for the opportunity to discuss the paper and for a terrific presentation.

Dr Griepp and the group at Mount Sinai have contributed enormously to our knowledge and understanding of spinal cord anatomy and pathophysiology over the years, and the group continues to make important observations in an effort to advance the field of thoracoabdominal repair, specifically regarding spinal cord protection and the prevention of the devastating complications of paraplegia.

With regards to your presentation, I have a couple of questions.

Animal models, although indispensable in our understanding of human pathology, have limitations. Spinal cord anatomy and its circulation, for example, differs greatly between pigs and humans. And your group has previously described these differences in great detail and have noted that the Yorkshire pigs, correcting for weight and body surface area, have much larger internal thoracic and subscapular arteries than the human, supplying extensive collateral blood flow to the lower body and consequently to the spinal cord. Pigs also have large bilateral vertebral arteries and smaller segmental, thoracic and lumbar arteries, and the aortic bifurcation is also quite different in the pig, with a median sacral artery arising as a large-caliber vessel approximating the size of the common iliac artery. Importantly, surgical ligation or coiling of the segmental arteries followed by delayed TEVAR is not a perfect replication of an open extensive thoracoabdominal aneurysm replacement and specifically the Crawford extent II repairs.

My question to you is, how would these differences go into the interpretation of your results? Further, how did the authors choose a 7- to 10-day time frame for the coil embolization? And how would the segmental arteries, which were coiled, which varied between the groups, how exactly were they chosen? And is there really an upper limit?

The other interesting thing would be that the reduction of histologic spinal cord damage was most prominent to the area in which segmental arteries were coiled. This implies a delicate balance of an ischemic stimulus leading to protective angiogenesis without immediate necrosis or permanent injury. How could you hypothesize about the specific protective factors that are activated by the limited hypoxia during coil embolization for segmental arteries? I agree with you entirely that the approach is novel and certainly worthy of a clinical trial.

Dr Geisbüsch. Thank you, Dr Coselli, for your comments and your questions. I would like to first comment on the 7- to 10-day time frame we chose. As you know, we previously undertook studies in our laboratory where we studied what happened after 2-stage procedures. And for those procedures we chose a week in-between those staged repairs where we either surgically ligated the abdominal segmental arteries and the thoracic or through hybrid procedures, and we found that a week is enough, at least

in pigs, to stimulate some kind of vascular remodeling and that this improved outcome dramatically.

Through anatomical studies we also studied the pathophysiology and we could see that after the operation there is a drop in collateral network pressure as a percentage of MAP, which we could measure, and then this pressure increases again 24 hours after the operation and recovers within 5 days. This is how we hypothesize that this is a vulnerable time frame during which vascular remodeling takes place.

During previous studies in the laboratory at Mount Sinai, in which we tried to image the vasculature of the spinal cord, we could see that the vessels nourishing the spinal cord—especially the anterior spinal artery—increases in diameter over a time frame of 5 days, and that is how we decided to try 7 to 10 days. In patients, obviously, we cannot be sure whether this is an adequate interval and that is why we think we need to identify a way to clinically monitor the induced ischemia and vascular remodeling to find the optimal interval after the induced ischemic stimulus for extensive aneurysm repair in patients.

We also observed that development of collaterals as response to diminished input after SA sacrifice is most prominent in the lower thoracic/upper lumbar region, whereas the vessels at the caudal and cranial ends of the spinal cord, chiefly the anterior spinal artery, increase in size as an immediate response. We therefore chose to embolize vessels in the T11 to L3 region to stimulate angiogenesis and arteriogenesis in advance. In contrast to 2-stage procedures, where a large number of intercostal vessels are sacrificed, we sought to find a less invasive strategy by occluding only a small number of SAs, and we succeeded in showing that this is enough to stimulate this protective arteriogenic/angiogenic response.

Dr Coselli. Your group has previously reported on improved results with paraplegia with staged repairs for thoracoabdominal aneurysms, but this particular model may be more akin to TEVAR in patients who have previously had abdominal aortic aneurysm replacement.